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Response of Selected Hard Red Wheat Lines to Imazamox as Affected by Number and Location of Resistance Genes, Parental Background, and Growth Habit

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ABSTRACT

Imidazolinone-resistant (IR) wheat (*Triticum aestivum* L.) was released for commercial production in portions of the USA in 2002 and has provided growers with a new technology to selectively control winter annual grass weeds. Imidazolinone herbicides inhibit acetolactate synthase (ALS) in susceptible plants; however, IR wheat has an altered target site which confers resistance to these herbicides. The mutation-derived resistance trait of most commercially available IR winter wheat cultivars is located on the D-genome; however, winter and spring wheat cultivars with the resistance trait on the A, B, or D genome or on multiple genomes are currently under development. Four groups of near-isoline wheat with spring or winter growth habit and resistance genes on the B, D, or both B and D genomes were compared for whole plant and ALS enzyme response to imazamox. Biomass accumulation after treatment was similar among B- and D-genome resistant winter wheat biotypes and was always higher than B- and D-genome resistant spring wheat biotypes. D-genome resistant spring wheat was more resistant than B-genome resistant spring wheat and the two-gene resistant spring wheat had an additive level of tolerance to imazamox compared with single-gene resistant spring wheat. Growth habit (spring vs. winter) did not affect in vitro ALS activity among B- or among D-genome resistant cultivars; however, D-genome resistant cultivars had significantly higher in vitro ALS activity in the presence of imazamox compared with B-genome resistant cultivars regardless of growth habit. D-genome resistance appears to provide greater tolerance to imazamox compared with B-genome resistance; however, multiple-genome resistance likely will be required to consistently avoid crop injury in spring wheat from labeled U.S. rates. Although ALS extracted from winter wheat and spring wheat responded similarly to imazamox, whole plant responses demonstrates that tolerance is affected by factors other than resistance gene location.

HERBICIDE RESISTANT (HR) crop cultivars exist for most major world crops and several minor crops and ornamentals (Devine, 2005). Growers in North America, in general, quickly accepted HR crop technology with 70 to 80% market share in some crops (Duke, 2005). Imidazolinone-resistant (Clearfield) winter wheat was made commercially available in 2002 in the Great Plains and in 2003 in the Pacific Northwest regions of the USA. The imidazolinone herbicides inhibit acetolactate synthase (ALS, also known as acetohydroxyacid synthase, EC 4.1.3.18), the first enzyme unique to the biosynthesis of the branched-chain amino acids (Shaner et al., 1984). Imazamox (Beyond) was registered for use on IR-wheat because of the herbicides weed spectrum, limited soil persistence, and toxicological properties (Anonymous, 2004; Shaner et al., 1996). This technology has provided new opportunities for selective control of winter annual grass weeds including jointed goatgrass (*Aegilops cylindrica* Host), feral rye (*Secale cereale* L.), Italian ryegrass (*Lolium multiflorum* Lam.), and the brome complex (*Bromus* spp.) in wheat (Geier et al., 2004; Zemetra et al., 1998).

The IR trait (FS4) was isolated after wheat-seed mutagenesis and screening with imazethapyr {1-[(2-chloro-5-pyridyl)methyl]-*N*-nitro-imidazolidin-2-imine}. Controlled crossing followed by progeny testing with herbicide treatment indicated that one gene provided tolerance to the imidazolinone herbicides (Newhouse et al., 1992). The original wheat mutant and most currently commercialized wheat cultivars have the resistant allele on the long arm of chromosome six in the D genome (Anderson et al., 2004). Because modern wheat is an allohexaploid ($2n = 42$) consisting of three diploid genomes: A, B, and D (Poehlman and Sleper, 1995) and ALS in wheat is produced by a multigene family, the enzyme produced by the A and B genome homoeologues remains susceptible to imidazolinone herbicides (Pozniak et al., 2004a). New IR-wheat cultivars are under development using conventional breeding techniques and existing mutations or a combination of seed mutagenesis and conventional breeding (Tan et al., 2005). These efforts have resulted in mutation-derived wheat lines with the imidazolinone resistance trait on the long arm of chromosomes 6D, 6B, and 6A (*AhasL-D1*, *AhasL-B1*, and *AhasL-A1*) (Pozniak et al., 2004a). Conventional backcrossing programs are being used to create wheat lines with multiple-genome imidazolinone resistance.

While IR-winter wheat generally has adequate safety to labeled rates of imazamox [(RS)-2-(4-isopropyl-4-methyl-5-oxo-2-imidazolin-2-yl)-5-methoxymethylnicotinic acid], crop injury occasionally occurs (Geier et al., 2004). IR-spring wheat appears to be more sensitive to imazamox than winter wheat and single-gene resistant cultivars do not have adequate tolerance to U.S. labeled rates of the herbicide (Pozniak et al., 2004b, Tan et al., 2005). Wheat injury consists of minor chlorosis, dark green color of developed leaves, or stunting and yield loss can occur (Pozniak et al., 2004b). Crop injury can be influenced by application parameters such as high imazamox rates, surfactants, and application timing (Frihauf et al., 2005; Geier et al., 2004; Stougaard et al., 2004). Anecdotal evidence also suggests that

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Abbreviations: ALS, acetolactate synthase; HR, herbicide resistant; IR, imidazolinone resistant.

environmental stresses during or immediately following imazamox application may influence injury (M. Dahmer, personal communication).

Wheat injury potential may differ for wheat cultivars that are currently in development. Most commercially available cultivars have the D-genome resistance gene; however, both spring and winter wheat cultivars are being developed in which the resistant ALS gene is located on the A, B, or D genome, or on multiple genomes. Although several new cultivars are nearing commercialization in North America, relatively little data exist on the relative imazamox sensitivity of winter and spring wheat, B- vs. D-genome resistance, or on single-gene vs. two-gene resistance in hard red wheat.

The objectives of this research were to quantify and contrast the imazamox sensitivity of several hard, red, IR-wheat near-isolines with different parental backgrounds, resistance gene locations, and growth habit at the whole plant and ALS-enzyme level.

MATERIALS AND METHODS

Wheat Seed Source

Seed for the selected hard red winter wheat lines were obtained from their respective public or private breeders. Wheat lines used in the experiments included both commercially available cultivars and advanced breeding selections. The specific lines used in the experiments included four groups of near isolines of wheat based on the hard red spring cultivars Gunner and Teal and on the hard red winter cultivars Millennium and Wahoo (Table 1) (Baenziger et al., 2001, 2002; Hughes and Hucl 1993). Each of the groups included a susceptible parent, a line with resistance on the B genome, and a line with resistance on the D genome. Because we did not have access to a D-genome resistant line derived from Teal, the D-genome resistant line BW755 (Grandin by FS4) was included in the Teal group instead. Additionally, three two-gene (BD) resistant lines were included in the spring wheat groups, two with Gunner parentage and one with Teal parentage. Each of the IR-wheat lines used in the experiment carried the same mutation resulting in a serine to asparagine substitution at amino acid residue 653 relative to the *Arabidopsis thaliana* (L.) Huynh. consensus sequence (M. Dahmer, personal communication). The currently available imidazolinone-resistant winter wheat cultivar Above (D-genome resistance, 'TAM110' background) also was included in all experiments as a standard (Haley et al., 2003; Lazar et al., 1997).

Table 1. Hard red wheat lines used in imazamox dose–response experiments.

Line	Type	Resistance†	Parental background
Gunner	spring	Susc.	Gunner
AP601CL	spring	D	Gunner
AP602CL	spring	B	Gunner
AP603CL	spring	BD	Gunner
AP604CL	spring	BD	Gunner
Teal	spring	Susc.	Teal
BW755‡	spring	D	Grandin
Teal11A	spring	B	Teal
HRS2G	spring	BD	Teal
Wahoo	winter	Susc.	Wahoo
WAH001	winter	D	Wahoo
WAH040	winter	D	Wahoo
WAH002	winter	B	Wahoo
WAH005	winter	B	Wahoo
Millennium	winter	Susc.	Millennium
MIL041	winter	D	Millennium
MIL051	winter	D	Millennium
MIL002	winter	B	Millennium
MIL009	winter	B	Millennium
Above	winter	D	TAM110

† Susc., D, B, and BD indicate ALS susceptible wheat, D-genome resistance, B-genome resistance, and double gene (B- and D-genome) resistance, respectively.

‡ BW755 was used in the experiments because a Teal derived line with D-genome resistance was unavailable.

Whole Plant Dose–Response Experiments

Four wheat seeds were planted in commercial potting media[1] in 12-by-12-by-8-cm pots[2]. Eight pots of an individual wheat line were placed in a 25-by-50-cm flat. Plants were grown in a greenhouse under natural light conditions supplemented with 400 W sodium halide lamps to provide a 14 h daylength. Day/night temperatures were 24 and 18°C, respectively, and the plants were irrigated with an automatic sprinkler system. At the 2.5 to 3 leaf (lf) stage, the eight pots in a flat were randomly assigned treatments consisting of six imazamox doses (1.3, 3.9, 11.7, 35, 105, 315 g ai/ha), an untreated control, and a zero-time biomass determination. Recommended field rates of imazamox range from 35 to 53 g/ha in winter wheat grown in the USA (Anonymous, 2004). Imazamox treatments were applied with a cabinet sprayer calibrated to deliver 145 L/ha and included 0.25% (v/v) nonionic surfactant (NIS) and 2.5% (v/v) urea ammonium nitrate (UAN). On the day of herbicide application, wheat plants in one of the untreated pots from each flat were clipped at the soil surface, fresh weight was measured, and the plants were dried in a 60°C oven for 24 h for zero-time dry weight determination. All treated plants and the remaining untreated plants were returned to the greenhouse and maintained for 21 d after imazamox treatment (DAT). Biomass from all remaining plants was harvested 21 DAT for fresh and dry weight measurements.

Each group of wheat lines (i.e., those with a common parent) along with Above as a standard was examined in a separate experiment. All experiments were arranged as a randomized complete block design with a full factorial (wheat line × imazamox rate) arrangement of treatments and four replications. Each experiment was repeated. Biomass data from each wheat cultivar were converted to biomass accumulation after application by subtracting the appropriate average zero-time biomass from each sample. Dry biomass data were converted to a percentage of the untreated control plants within each cultivar to allow direct comparisons between groups. Generalized nonlinear (Proc NLMIXED, SAS 2001) regression analyses assuming a normal distribution were employed to estimate a log-logistic model (Seefeldt et al., 1995) given by:

$$y = C + \frac{D - C}{1 + (x/GR_{50})^b} \quad [1]$$

In this equation, y = dry biomass accumulation (expressed as a percent of the control), x = imazamox rate, C = lower response limit, D = upper response limit, b is a rate parameter related to response to increasing imazamox dose, and GR_{50} is the imazamox rate that caused a 50% reduction in biomass accumulation. Adequacy of model fit was determined by testing for the significance of regression coefficients, evaluating correlation among parameters, and examining the underlying residual structure.

[1] Metro-Mix 200 potting mix, Sun Gro Horticulture, Inc., 15831 NE eighth St, Suite 100, Bellevue, WA 98008.

[2] 801 True series inserts, ITML Horticultural Products, Inc., 75 Plant Farm Blvd., PO Box 265, Brantford, ON, Canada. N3T 5MB.

Comparisons of regression lines and examination of parameter estimate confidence intervals were used to determine if wheat cultivars could be combined into spring and winter types and/or B, D, and BD resistance groups. Single degree of freedom contrasts were used to determine differences between parameter estimates among the wheat types.

In Vitro Acetolactate Synthase Dose–Response Experiments

Acetolactate synthase extraction and measurement of activity were performed on stem tissue from 3-leaf wheat plants by procedures modified from Pozniak et al. (2004a) and Singh et al. (1988). Wheat plants from each cultivar (Table 1) were seeded in commercial potting mix in 25-by-50-by-8-cm flats (approx. 50 per flat) and grown in a greenhouse under previously described conditions. Assay reactions were performed in a 100-μL volume in 96-well microtiter plates. Imazamox was diluted such that final concentrations in the assay reactions were 0.2, 0.4, 0.8, 1.6, 3.1, 6.3, 12.5, 25, 50, and 100 μ*M*. Other test solutions in the microtiter plates included distilled water (control), 100 μ*M* ACC299,016 [inhibits all forms of ALS enzyme (Alvarado et al., 1992)], and distilled water plus 5% sulfuric acid (stops all enzymatic activity). Crude extracts were used in the assay by adding 50 μL of extract supernatant to wells containing 50 μL of test solution.

Acetolactate production was determined by decarboxylating acetolactate to acetoin and measuring absorbance spectrophotometrically at 535 nm according to the methods of Westerfeld (1945). Acetolactate production data were expressed as ALS activity as a percentage of the mean of the no-imazamox controls minus the background absorbance in the ACC299,016 treatment. Each group of wheat lines (i.e., those with a common parent) was examined in a separate experiment for logistical reasons; however, relevant comparisons among wheat lines were made using the relative ALS activity data. Each imazamox dose range was replicated four times within an experiment and each experiment was repeated. Mean activity from the imazamox concentration that completely inhibited the susceptible wheat was compared using Fisher's protected LSD with an α level of 0.05. When the ALS activity in sensitive wheat plants was completely inhibited by an in vitro imazamox dose, it was assumed that the sensitive form of the enzyme in the resistant cultivars also was inhibited—leaving only the resistant form of the enzyme active.

RESULTS

Whole Plant Dose–Response Experiments

Analysis of variance indicated no effect of experimental replication on the results of the dose–response experiments; therefore, data from two experiments for each parental group were combined for further analysis. Examination of the dose–response curves and parameter estimates for individual wheat cultivars indicated strong similarities among cultivars with the same type of resistance and growth habit (data not shown). Above wheat responded consistently in all experiments and was similar to the other winter, D-genome resistant wheat cultivars. The individual cultivars were combined into growth habit/resistance type groups (Spring Susc., B, D, and BD; Winter Susc., B, and D, respectively) for further analysis. Above was not combined with the other winter D-genome lines for analysis.

The log-logistic model accurately described biomass accumulation after imazamox application for susceptible and resistant wheat types (Fig. 1). Because biomass data were expressed as a percentage of the untreated control plants within cultivar and experiment, the upper and lower limit parameters, D and C were held at 100 and 0%, respectively. The GR_{50} parameter and the rate parameter, b, were allowed to vary among groups and ranged from 1.6 to 325.6 and −0.8 to −1.8, respectively (Table 2). The GR_{50} and *b* parameters were significant for all wheat groups (Table 2) and residual analysis indicated that the errors generally conformed to the underlying regression assumptions (data not shown).

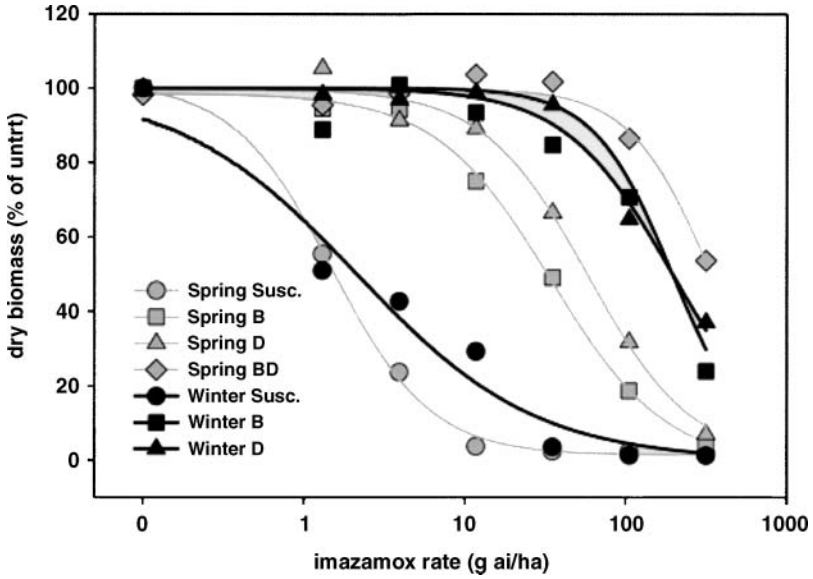

Fig. 1. Effect of foliar applied imazamox dose on wheat biomass accumulation 21 d after treatment (DAT) for seven wheat types. Mean dry biomass data (% of untreated) and predicted values are represented by the gray symbols and lines and black symbols and lines for the spring and winter wheat types, respectively.

Table 2. Model parameter estimates, standard errors, and *P* values estimated by log-logistic regression for biomass accumulation by seven wheat classes and Above wheat in response to foliar imazamox treatment. † ‡

Wheat class§	Parameter	Estimate	Standard error	P-value
Spring susc.	GR_{50}	1.57	0.27	<0.0001
	b	−1.37	0.33	<0.0001
Spring B	GR_{50}	31.87	5.20	<0.0001
	b	−1.20	0.21	<0.0001
Spring D	GR_{50}	57.43	8.92	<0.0001
	b	−1.33	0.25	<0.0001
Spring BD	GR_{50}	325.59	39.78	<0.0001
	b	−1.78	0.45	<0.0001
Winter susc.	GR_{50}	2.14	0.49	<0.0001
	b	−0.78	0.12	<0.0001
Winter B	GR_{50}	161.84	16.61	<0.0001
	b	−1.46	0.26	<0.0001
Winter D	GR_{50}	196.41	23.75	<0.0001
	b	−1.29	0.20	<0.0001
Above	GR_{50}	195.95	22.15	<0.0001
	b	−1.78	0.32	<0.0001

† The upper limit parameter, D, was set to 100% and the lower limit, C, was set to 0% for all wheat types.
‡ Abbreviations: C, lower response limit; D, upper response limit; *b*, a rate-related parameter; GR_{50}, imazamox treatment (g ai/ha) that caused a 50% reduction in biomass accumulation.
§ Spring and Winter indicate growth habit and susc, B, D, and BD indicate ALS susceptible, B-genome resistance, D-genome resistance, and double gene resistance, respectively. Above is a widely grown D-genome resistant winter wheat included as a standard in each experiment.

Contrasts of biomass data indicated that the rate parameter, *b*, did not differ among any comparisons of B- and D-genome resistance with spring or winter growth habit (Table 3). These results are not surprising since others have proposed that parallel dose–response curves suggest a similar mechanism of resistance (i.e. all have site-of-action based resistance) (Seefeldt et al., 1995). Contrasts of the GR_{50} parameter indicated no difference between B and D genome resistance in a winter wheat background ($P = 0.2331$), but there were differences between resistance types in a spring wheat background ($P = 0.0135$). There were differences in GR_{50} values between spring and winter B-genome resistance ($P < 0.0001$) and between spring and winter D-genome resistance ($P < 0.0001$). Two-gene resistant spring wheat had a higher GR_{50} value than the single-gene resistant spring wheat groups ($P < 0.0001$).

Table 3. Contrasts between GR_{50} and *b* parameters for biomass accumulation by seven wheat classes in response to foliar imazamox treatment.

Contrast†	Parameter‡	*F* value	*P* value
Spring B vs. spring D	GR_{50}	6.13	0.0135
	b	0.16	0.6882
Winter B vs. winter D	GR_{50}	1.42	0.2331
	b	0.26	0.6098
Spring B vs. winter B	GR_{50}	55.77	<0.0001
	b	0.61	0.4360
Spring D vs. winter D	GR_{50}	30.02	<0.0001
	b	0.01	0.9061
Spring single-gene vs. spring two-gene§	GR_{50}	80.35	<0.0001
	b	1.17	0.2794

† Spring and Winter indicate growth habit and susc, B, D, and BD indicate ALS susceptible, B-genome resistance, D-genome resistance, and double gene resistance, respectively.
‡ Abbreviations: *b*, a rate-related parameter; GR_{50}, imazamox treatment (g ai/ha) that caused a 50% reduction in biomass accumulation.
§ Single-gene spring wheat included both B- and D-genome resistance wheat types and spring two-gene included three two-gene cultivars.

In Vitro Dose–Response Experiments

Analysis of variance indicated no effect of experimental replication (data not shown); therefore, data were combined for analysis. ALS extracted from wheat cultivars with similar growth habit (winter vs. spring) and resistance gene location (B- vs. D- vs. B- and D-genome) responded similarly to imazamox and were grouped into seven growth habit/resistance type groups as previously described. Above wheat responded consistently in all

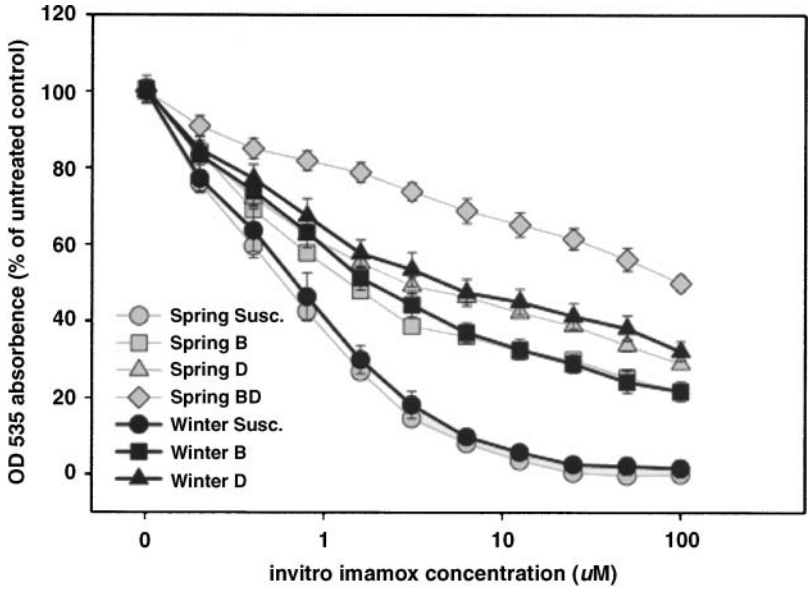

Fig. 2. Effect of imazamox on in vitro ALS activity of crude extract from seven wheat types expressed as a percentage of the untreated control wells. Mean ALS activity of spring and winter wheat are represented by gray symbols and lines and black symbols and lines, respectively. Error bars represent 95% confidence intervals of the mean ALS activity.

Table 4. Effects of 50 μ*M*† imazamox in vitro on acetolactate synthase (ALS) activity of seven wheat classes and Above wheat. ALS activity is expressed as a percentage of the untreated control wells within a wheat cultivar and experiment.

Wheat Class‡	Mean ALS activity (% of untrt)	Standard error	95% confidence interval
Spring susc.	−0.3	0.6	1.2
Spring B	25.4	1.0	2.1
Spring D	33.8	0.7	1.6
Spring BD	56.1	1.4	3.0
Winter susc.	2.2	1.1	2.3
Winter B	25.0	1.4	2.9
Winter D	37.6	1.6	3.3
Above	39.2	2.0	3.9
$LSD_{(0.05)}$	3.9	–	–

† The 50-μM imazamox concentration was selected for analysis because ALS activity of both susceptible wheat types was completely inhibited. The susceptible form of the ALS enzyme was also assumed inhibited in the resistant wheat types leaving only the resistant form of the enzyme active.

‡ Spring and Winter indicate growth habit and susc., B, D, and BD indicate ALS susceptible, B-genome resistance, D-genome resistance, and double-gene resistance, respectively. Above is a widely grown D-genome resistant winter wheat included as a standard in each experiment.

experiments and was similar to the other winter, D-genome resistant wheat cultivars but was not included in the winter, D-genome group.

There was a significant response to increasing imazamox concentration for all wheat types (Fig. 2). In vitro ALS activity data, however, did not fit a sigmoidal response curve. Because the resistant wheat cultivars in these experiments have both susceptible and resistant forms of the ALS enzyme, the lack of sigmoidal response probably is due to overlapping response curves for the two forms of the enzyme and the relatively low imazamox concentrations (C. Preston, personal communication). To make comparisons between ALS activities from different wheat groups, Fisher's protected LSD tests were used on data from the 50-μ*M* imazamox concentration. This concentration was the lowest imazamox dose that completely suppressed in vitro ALS activity in extracts from susceptible wheat, and it was assumed that all susceptible ALS in resistant lines was completely inhibited at this concentration.

There were differences between in vitro ALS activity in biotypes with B- and D-genome resistance. D-genome resistant wheat had 25 and 33% more ALS activity in the spring and winter wheat types, respectively, compared with B-genome resistant wheat in 50μM imazamox (Table 4). Two-gene resistant spring wheat had 40 to 55% higher ALS activity than B- or D-genome resistant spring wheat.

DISCUSSION

While results from greenhouse screening may not correspond directly with field observations, combined data from the whole plant assays and in vitro enzyme assays provide some interesting observations about IR-wheat. First, under greenhouse conditions, growth of both winter and spring IR-wheat was reduced with commonly used field rates of imazamox (predicted 4 to 52% reduction in biomass accumulation at 35 g ai/ha imazamox) during the time course of these experiments. Field experiences suggest that early biomass reductions may not persist throughout a typical field season. Second, whole plant response to imazamox differed among winter and spring wheat. IR-winter wheat was much more resistant to imazamox than IR-spring wheat regardless of resistance gene location. Additionally, there were differences in biomass accumulation between B- and D-genome resistance in spring wheat but not winter wheat at the rates tested. Within IR-spring wheat, D-genome resistant cultivars were more resistant than cultivars with B-genome resistance while in winter wheat B- and D-genome resistant cultivars had similar biomass accumulation after imazamox application.

Differences in whole plant response can be partially explained by the in vitro ALS response to imazamox. When exposed to 50 μM imazamox, only 25.2% of the total in vitro ALS activity remained in the B-genome wheat extract compared with 35.7% remaining in the D-genome wheat extract, suggesting that the D genome accounts for more of the total ALS activity in wheat. While there were differences in ALS activity between B- and D-genome resistance in both spring and winter wheat, there was no corresponding difference in whole plant biomass response in the winter wheat groups. This suggests that factors other than genome location (such as higher rates of herbicide metabolism) may influence the tolerance of winter wheat to imazamox.

This research clearly demonstrates that the number and location of resistance genes as well as the wheat's growth habit affect the response of hard red IR wheat to imazamox. Spring wheat lines with resistance on both the B- and D-genomes had an apparently additive increase in ALS activity in vitro and a 6- to 10-fold increase in whole plant GR_{50}, which is similar to other published reports (Pozniak and Hucl, 2004; Pozniak et al., 2004a; Rainbolt et al., 2005). Although we hypothesize that a similar increase in resistance is likely for two-gene winter wheat, no two-gene winter wheat lines were available for comparison in these experiments. In spring wheat, two resistance genes will likely be necessary for adequate (2×) crop safety to U.S. labeled imazamox rates. Two-gene resistance or resistance on all three wheat genomes will provide maximum crop safety in both winter and spring IR wheat systems.

Genome location of the resistance gene strongly affected in vitro ALS activity possibly because of unequal production of the enzyme among the three genomes. In these experiments, the D genome wheat lines appeared to have 10% more of the total in vitro ALS activity compared with B-genome, which is consistent with Pozniak et al. (2004a). The B- vs. D-genome in vitro response was similar for winter and spring wheat lines although whole plant differences were only observed in spring wheat lines. Differences among ALS produced by the multiple genomes in wheat have implications for IR wheat cultivar development. If single-gene or two-gene resistance is desired, higher levels of imidazolinone resistance may result from parents with D-genome resistance compared with B-genome parents. Selection of parents with the D-genome resistance could result in winter

wheat cultivars with adequate field tolerance to imazamox while requiring fewer crosses and backcross generations. Alternatively, selection of IR parent lines with A- and/or B- genome resistance may reduce the potential for pollen-mediated gene flow to jointed goatgrass, which shares the D-genome with wheat (Hanson et al., 2005; Zemetra et al., 1998). The apparent differences in ALS production between the B- and D-genomes may also support hypotheses that the D-genome is a more recent addition to polyploid wheat than the A or B genomes and as such has a greater impact on the plants metabolic processes (Kimber and Sears, 1987).

Parental background did not appear to significantly affect biomass accumulation or ALS activity on the basis of these limited data; however, others have suggested that parental background or market class may affect wheat ALS sensitivity to imazamox (Rainbolt et al., 2005). Growth habit (winter vs. spring) did not affect ALS activity in vitro; however, biomass accumulation after imazamox application was considerably higher in all winter wheat lines compared with spring lines with similar resistance genes. This difference has been noted anecdotally (M. Dahmer, personal communication), but has not been experimentally investigated. Potential factors resulting in greater imidazolinone resistance in winter wheat may include differences in imazamox metabolism, absorption, or translocation.

Future research in hard red wheat should address the cause of greater resistance in winter wheat and also on the relative contribution of each of the genomes to total resistant ALS production (alone and in various two-gene combinations) in winter and spring wheat lines. Expression of ALS genes may differ among parental lines, market classes, or growth habit and is a promising area of research. Finally, the effects of environmental stress alone or in combination with various wheat genotypes may provide further information on IR wheat resistance to imazamox.

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